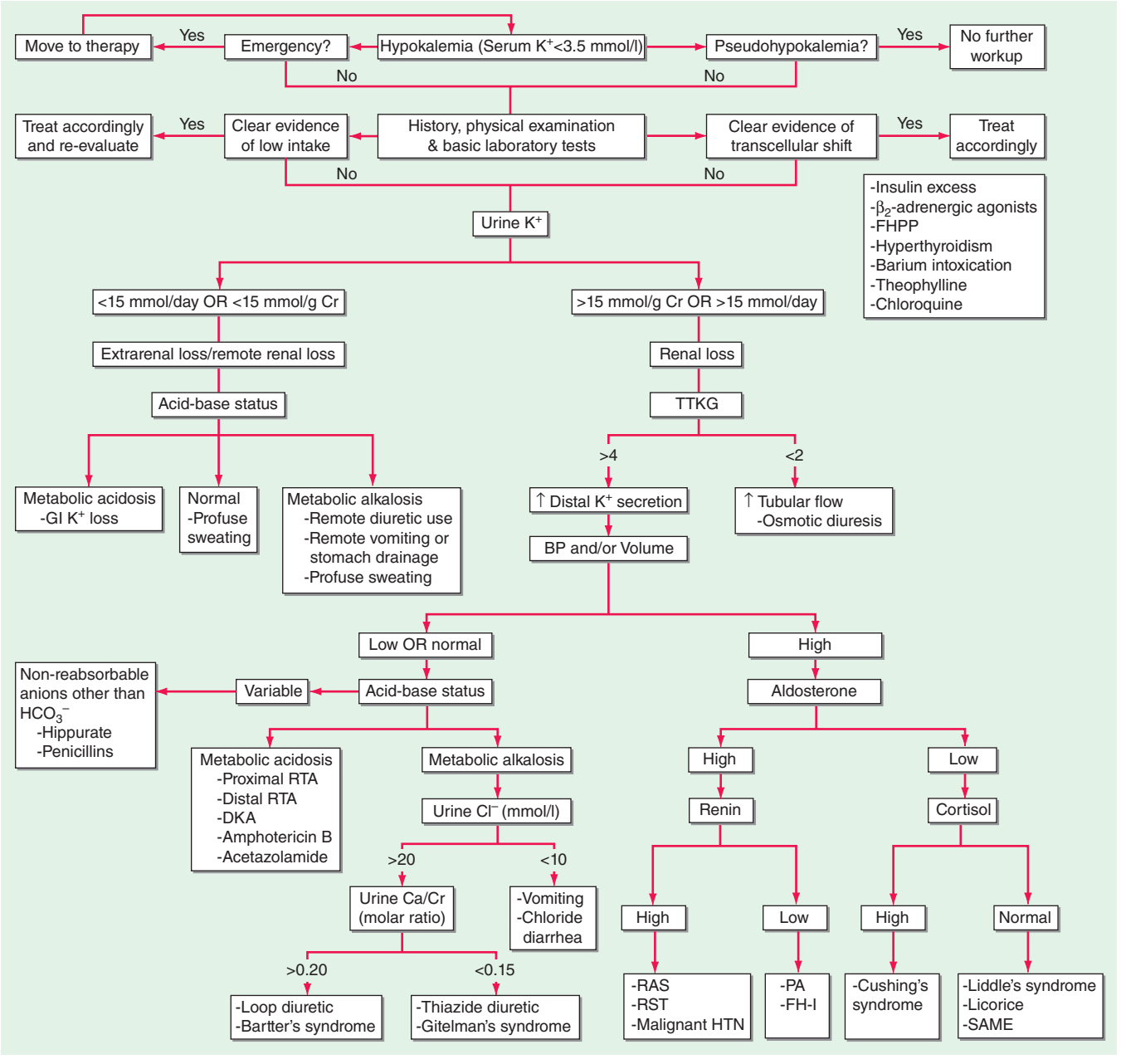

FIGURE 63-7 The diagnostic approach to hypokalemia. See text for details. AME, apparent mineralocorticoid excess; BP, blood pressure; CCD, cortical collecting duct; DKA, diabetic ketoacidosis; FH-I, familial hyperaldosteronism type I; FHPP, familial hypokalemic periodic paralysis; GI, gastrointestinal; GRA, glucocorticoid remediable aldosteronism; HTN, hypertension; PA, primary aldosteronism; RAS, renal artery stenosis; RST, renin-secreting tumor; RTA, renal tubular acidosis; SAME, syndrome of apparent mineralocorticoid excess; TTKG, transtubular potassium gradient. *(Used with permission from DB Mount, K Zandi-Nejad K: Disorders of potassium balance, in Brenner and Rector's The Kidney, 8th ed, BM Brenner [ed]. Philadelphia, W.B. Saunders & Company, 2008, pp 547-587.)*

exacerbate hypokalemia. The peripheral intravenous dose is usually 20–40 mmol of K^+-Cl^- per liter; higher concentrations can cause localized pain from chemical phlebitis, irritation, and sclerosis. If hypokalemia is severe (<2.5 mmol/L) and/or critically symptomatic, intravenous K^+-Cl^- can be administered through a central vein with cardiac monitoring in an intensive care setting, at rates of 10–20 mmol/h; higher rates should be reserved for acutely life-threatening complications. The absolute amount of administered K^+ should be restricted (e.g., 20 mmol in 100 mL of saline solution) to prevent inadvertent infusion of a large dose. Femoral veins are preferable, because infusion through internal jugular or subclavian central lines can acutely increase the local concentration of K^+ and affect cardiac conduction.

Strategies to minimize K^+ losses should also be considered. These measures may include minimizing the dose of non-K^+-sparing diuretics, restricting Na^+ intake, and using clinically appropriate combinations of non-K^+-sparing and K^+-sparing medications (e.g., loop diuretics with angiotensin-converting enzyme inhibitors).

HYPERKALEMIA

Hyperkalemia is defined as a plasma potassium level of 5.5 m*M*, occurring in up to 10% of hospitalized patients; severe hyperkalemia (>6.0 m*M*) occurs in approximately 1%, with a significantly increased risk of mortality. Although redistribution and reduced tissue uptake can acutely cause hyperkalemia, a decrease in renal K^+ excretion is the most frequent underlying cause (Table 63-5). Excessive intake of K^+ is a rare cause, given the adaptive capacity to increase renal secretion; however, dietary intake can have a major effect in susceptible patients, e.g., diabetics with hyporeninemic hypoaldosteronism and chronic kidney disease. Drugs that impact on the renin-angiotensin-aldosterone axis are also a major cause of hyperkalemia.